Exposure to parental stress increases pollution-related lung damage in children

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Psychosocial stress appears to enhance the lung-damaging effects of traffic-related pollution (TRP) in children, according to new research from the Keck School of Medicine of the University of Southern California (USC) in Los Angeles.

The results will appear online ahead of the print edition of the American Thoracic Society's American Journal of Respiratory and Critical Care Medicine.

"This is the first study demonstrating that growing up in a stressful household was associated with larger traffic pollution-induced lung deficits in healthy children compared to low stress households," said lead researcher Talat Islam, MBBS, assistant professor in the Division of Environmental Health at Keck School of Medicine.

Dr. Islam and colleagues administered a validated stress questionnaire, the Perceived Stress Scale (PSS), to the parents of nearly 1,400 children who participated in the USC Children's Health Study in Southern California. The children, ranging between 10-12 years old, were assessed for lung function and other respiratory health outcomes. Their exposure to traffic-related pollutants (TRP) was assessed by estimating exposure to nitric oxide (NO), nitrogen dioxide (NO2), and total oxides of nitrogen (NOX) at school and at home.

The researchers identified a number of socio-demographic factors associated with stress levels among the participants' parents. Hispanic

and Asian parents had relatively higher levels of perceived stress than White parents. Characteristics associated with low socioeconomic levels were also associated with high stress, such as income below $30,000 a year, low parental education, lack of health insurance and lack of an air conditioner in the home. Similarly, exposures to traffic-related pollution varied widely within the study. NOX, for example, ranged from 6 to 108 ppb in different locations.

The researchers did not observe any statistically significant associations between parental stress alone and lung function levels in children. However, they found that as levels of traffic-related pollution increased among children who grew up in high-stress households, lung function decreased, but there was no corresponding lung effect in low- stress households. In high stress households, children had on average a 4.8 percent and 4.5 percent lower lung volume (FVC) and flow in the larger airways (FEV1) for each 22 ppb increase in NOX. "Based on the emerging data we expected to see a modifying effect of stress," said Dr. Islam. "However, we were surprised by the magnitude of effect."

The study also revealed the novel finding that lung function declines were related to both at-home and at-school exposures. "Children in this age group spend almost one-third of their day-time hours at school so exposure at school is an important contributor to total exposure," said Dr. Islam. "Perhaps children maintain the chronic and systemic effect of stress from their home environments as they go to school, further modifying their response to traffic exposure."

"One possible explanation for the stress-related pattern of TRP respiratory effects is the biological pathways common to effects of TRP and stress," said Dr. Islam. "Like air pollution, stress has been linked to both inflammation and oxidative damage at the cellular level, so this may explain the association."

While further research is needed on biological pathways, Dr. Islam believes the public health implications are clear: "The magnitude of the TRP-associated deficits in FEV1 and FVC levels in children growing up in high-stress households was larger than deficits reported for children exposed to maternal smoking during pregnancy and second-hand tobacco smoke," said Dr. Islam. "Our findings suggest that by regulating TRP levels around residential areas and schools, we could reduce the adverse effect of TRP on lung function among vulnerable children."

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